OBSCURE REFLEX SYMPTOMS OF TEETH, TONSILS AND SINUSES, ESPECIALLY THE SINUSES.

By ROBERT B. SWEET, M. D., Long Beach.

Since the advent of the era of the study of focal infections we have learned to consider lesions of the teeth, tonsils and sinuses collectively. We have learned that they have a common bacteriology and that they can and do produce remote infections in almost any tissue or organ in the body. But there is a tendency to look for gross lesions. By the aid of transillumination and the X-ray we search for the darkened areas of the sinuses and the indications of apical lesions of the teeth, often failing to find the more obscure lesions which may not be revealed even by our modern mechanical methods. It is to these obscure lesions which produce marked and intense reflex pain that I wish to direct your attention. In taking up the consideration of the pains and aches produced by lesions of the teeth, tonsils and sinuses, we discover that we have to deal with the common innervations of these structures through the branches of the fifth nerve, which enables us to understand the intricate manner in which they are connected. It is not the aching, decayed tooth; the sinus discharging pus; the hypertrophied turbinates; the impinging deflected septum; or the bulging bullaethmoidalis, so easily discerned by the casual observation that gives us the greatest trouble. It is those obscure lesions which produce much pain that tax the diagnostic acumen of the modern specialist.

Of the familiar reflexes produced by the teeth we well know the facial neuralgias and the lancinating pains, often remote from the site of the offending member. The pains in the eye, the congested conjunctival vessels, the supraorbital pains and aches in and about the ear. The earache caused by an impacted third molar is not an infrequent occurrence.

In looking about for perhaps the most obscure lesion of the teeth producing the greatest amount of disturbance, I have selected the dental pulp nodules. Not being well enough informed upon this subject personally, I have abstracted an article by Norman and Johnson (New York Medical Journal, July 20, 1921), that this paper may be more comprehensive in its scope; and to bring to the attention of the eye, ear, nose and throat physicians this phase of fifth nerve disturbances that should be given consideration in our efforts to solve some of the perplexing problems incident to fifth nerve neuralgia.

Neuralgia, defined in its primary sense, means pain, or a painful sensation of a definite type, limited to the sensory distribution of a peripheral nerve, or nerves. This pain may be referred to the surface distribution of the affected nerve, pressure on the areas of which causes a typical but not severe pain, which we are pleased to term neuralgic in character. In treating the neuralgias, our therapeutic resources are directed to a temporary or permanent relief of the chief symptom, pain. The causes of trigeminal neuralgia are many, and are mentioned for purposes of review as follows: The teeth play a predominant role, perhaps undergoing a constructive or destructive pathological change, or because of a constitutional defect in shape, position or development; affections of the gums, of the jaw bones, the cranial sinuses, and the mastoid, frequently produce neuralgias of the fifth nerve and associated neuralgias that are difficult to differentiate from a regional point of view; diseases of the eye, as iritis, cyclitis, iridocyclitis; diseases of the ear; constitutional diseases, as arteriosclerosis, malaria, anemias, diabetes mellitus (this disease being particularly prone to attack the teeth and gums), syphilis; the exogenous and endogenous intoxications; non-progressive convalescent states following acute infections and surgical procedure with pyogenic complications; the metabolic diseases with their associated endocrine disturbances; tumors of the Gasserian ganglion, of the brain, of bone in the adjacent vicinity or inflammation of the bony structures within the neighborhood of the nerve. Indeed, it may at times be the initial symptom of tabes dorsalis, and Oppenheim cites a case of multiple sclerosis beginning as a facial neuralgia. It is the purpose of this article to emphasize the necessity of excluding the presence of pulp nodules in apparently normal teeth, in all cases of intractable neuralgia, before one hopelessly resigns oneself to a state of helplessness in treating these cases. Pulp nodules in apparently sound and healthy teeth, with little or no reaction to the ordinary tests, are difficult of diagnosis. Pulp nodules, present in decaying teeth, are comparatively easy to detect, and their removal is but part of the operation, extraction or devitalization deemed necessary by the operator for that particular tooth.

Pulp Nodules—Pulp nodules are small masses of calcic material suspended in the pulp substance which, by reason of their progressive formation, effects a mechanical displacement to the point of strangulation, with resultant death of that tissue. They are commonly found in the bulb portion of the pulp, but may occur in the root portion, assuming a number of forms, those in the bulbar portion being round or nodular, while those in the root portion, are fusiform or spindle-shaped. They are more frequently mutiple than single, and it is believed that the larger nodules are formed by the coalescence of the smaller ones.

Symptomatology of Pulp Nodules—There appears to be no relationship between the frequency of occurrence of pulp nodules and the production of symptoms. They do not produce symptoms with any degree of consistency. They may be present and apparently do no harm, and, conversely, their presence may initiate an irritation of the terminal dental nerve filament because of mechanical pressure. It is a reasonable assumption that these nodules should produce symptoms in every case, but the converse is true in the majority of instances, paradoxical as it seems. This discrepancy between cause and effect may be accounted for by their slow formation, permitting the dental pulp to accommodate itself to the change within its tissue, without giving rise to symptoms. However, it appears that when the nodule or nodules become large enough to strangulate or obliterate the pulp tissue, symptoms are produced. The symptoms are the result of mechanical irritation, by the nodule or nodules, to the terminal nerve filament within the pulp tissue. Usually the local symptoms are hyperesthesias, not limited to the particular tooth or teeth affected, but affecting the pulps of all the teeth on that side of the dentures. Black has noted that general pulp hyperesthesia may be the precursor of an acute gouty or neuralgic attack. Unless the nodule or nodules are located and removed, continuous pain stimuli are transmitted by that branch of the fifth nerve supplying the affected tooth or teeth. It is known that if a sensory nerve transmits continuous pain stimuli, fatigue will result. Fatigue is the end result of biological changes, in nerve tissue, necessary for the performance of specific function. If fatigue is prolonged by reason of constant irritation (pain) to a terminal dental nerve filament, neuralgia of the main nerve

trunk results, with reflex neuralgic manifestations in its branches. This explains why the neuralgia does not disappear until some time after the local agent is removed. Time is necessary for a complete recuperation of the nerve itself.

So much for the teeth. As for the tonsils I have but passing comment to make. They have been given a great deal of consideration and their part in the production of pains and aches is quite generally understood. Probably the diagnosis of toxic neuritis covers the greater part of their etiological importance.

The sinuses constitute the most important and interesting division of the subject. I will not take time to enumerate the usual local pains about the antrum, the frontal headache, caused by acute sinusitis. The right maxillary sinusitis may manifest itself by a left supraorbital pain. The occipital and vertical pains and the pounding ears of an acute sphenoidal sinusitis are generally understood. In cases of acute sinusitis it is important not to be misled by the location of the pains, but the physician should seek by every method available to locate the offending sinus, and also make sure that it is only one, because several sinuses are frequently involved at the same time.

Your attention is directed to that class or type of sinus lesions which Sluder has classified in his book on "Headaches, and Eye Disorders of Nasal Origin." The cases showing small pathological changes but severe pain. Sluder's three classifications are the syndrome of nasal ganglion neurosis, vacuum frontal headaches, and hyperplastic sphenoiditis. Before I became acquainted with the contents of his book I was struck by the peculiar symptoms I had observed in a few cases that I had not remembered as being described in my text book. At least they did not correspond to the classical descriptions commonly taught. After becoming interested along this line I searched back through my records that I might ascertain how many of my cases I could place in this category. Of about four hundred cases of sinus trouble of which I had X-ray pictures and case records I was able to select only nine that I felt corresponded to one or the other of Sluder's classes. A clean-cut diagnosis of these conditions is not an easy task and usually the diagnosis is only confirmed by the results of the treatment. Take a case of recurrent intense headache extending over a long period of time often accompanied by vertigo and nausea; with the history of going to bed for three or four days, and finally resorting to a hypodermic of morphine for the relief of pain. When upon examination one finds the septum straight, the middle turbinate normal or slightly atrophied; no impinging areas; no pus or mucous or glarry secretion from any sinus, and transillumination and X-ray negative. And then upon opening of the nasal frontal duct have all the symptoms subside and the patient be relieved from that time on, one is warranted in making a diagnosis of vacuum frontal headaches.

Many of these patients have been seen by many specialists, who in the absence of any noticeable secretion have not made the diagnosis of sinusitis. Many of these patients have suffered for ten, twenty or even forty years and have been relieved by a single operation requiring less than twenty minutes' time. The cases of nasal ganglion neurosis and hyperplastic sphenoiditis are even more obscure, and no doubt if physicians were better trained in diagnosis and had more knowledge of the pathology of diseases of these areas, many more definitions describing more minutely these pathological lesions would be available.

I do not claim that ganglion neurosis and sphenoiditis are not caused by infection, or that they are always free from secretions. The point is that the secretion is not always in evidence. I have examined some patients time and again, finding the mucous membrane perfectly dry, but later on the tell-tale muco-purulent secretion would show up. I have always felt that the action of cocaine in shrinking the membrane acted not only to promote ventilation but also to facilitate the drainage of secretions. It is a fact also that the effect of negative suction or syphonage has had a most favorable effect upon these conditions. The Nichols nasal syphon used at home has a much better effect than the suction treatment in the office. After proper treatment the relief to these patients is so striking that it compensates for all the time and energy required to bring about favorable results.

CASE REPORTS

A man of 64 years of age had had headaches for the past eighteen years. He had consulted various specialists in several large cities, all of whom had suggested the possibility of sinus trouble but none had ever discovered enough evidence to warrant any operative procedure. He had had eight different prescriptions for glasses but was not wearing any of them. The attacks were periodic, occurring about every two weeks. They would begin with a slight dullness over the left eye, accompanied by gastric distress, nausea and ended usually in vomiting. He had long since given up hope of receiving any relief aside from opiates and a rest in bed for several days. His business was necessarily seriously interfered with. At no time during these attacks did he have any discharge from the nose. He was pale, his facial expression was distraught, thoroughly discouraged but willing to have anything done that would afford any hope of relief. He had two X-ray pictures that had been previously taken which showed nothing. But not being content with that I insisted on having another. This picture showed nothing. On examination the membranes of the nose were pale and slightly atrophic. The septum was straight. The middle turbinate hung free. There was no impingement, either against the septum or ethnoidal region. The bulla ethmoidallis was not prominent. There was no secretion, not even glarry mucous present, even after severe suction. The nasal frontal duct appeared patulous. Notwithstanding this lack of anything to warrant operative procedure, I proceeded to excenterate the anterior ethmoid cells, the agger-nasi, at the first sitting. Much to my surprise no pus or mucous was discovered, and only the usual amount of bleeding. This was followed by a marked improvement in the symptoms. The patient became enthusiastic over the prospect of other results. After that, whenever he felt an attack of headache coming on, he would come in, and with a shrinking up of the parts with cocaine and a slight curretage of the cells he would get relief. I became suspicious that the effects were either psychic or due to the soothing effects of the cocaine. Consequently, I frequently omitted the

cocaine and used adrenalin instead, with almost but not quite the same effect. The recurrence of attacks became less and less frequent. About this time, however, after I had felt that I had been dealing with a dry sinus, I began to notice the presence of a thick mucous which would flow down after the treatment and with the aid of suction. The patient also would tell me that he would often expectorate mucous an hour or two after the treatment. This fact has led me to question the existence of the so-called dry sinusitis.

Woman, age 32, three years ago began with periodic headaches, which gradually increased in intensity until they became severe enough to put her to bed for a period from three or four days to a week. She had been thoroughly examined and given a variety of treatments, including milk diet and rest, endrocrines and glandular extracts. But to no avail. Her eyes had been refracted. Sinuses and teeth had been examined by the X-ray. The sinuses had been examined by several physicians and pronounced negative. The patient's headaches were accompanied by vertigo, nausea, and even periods of delirium, often requiring opiates for relief. During the interval between attacks she was perfectly well and comfortable. On examination the nasal passages were entirely free from any signs of secretion, congestion or impingement. The possibility of frontal vacuum sinusitis was considered and a frontal sinus opened, but to no avail. At a secondary sitting the ethmoids were opened. Still no results. Finally, the left middle turbinate was removed, and the sphenoidal atrium exposed to view. There was a very small opening, hardly large enough to admit of a small probe. The orafice, however, presented a puckered appearance. The sphenoid was opened and the atrium enlarged. No discharge followed. This resulted in modifying the severity of the next attack very remarkably. Since then the attacks have become much less frequent and much less severe, until it has been possible to keep them entirely under control by local treatment of cocaine to the atrium, which is prone to close even after free opening has been made with a punch. There has never been any evidence of mucous or pus.

CONCLUSIONS

In searching for the cause of reflex pains of the fifth nerve and its connections through the sphenopalatine ganglion we should consider the teeth, tonsils and sinuses collectively.

The most intense reflex disturbances are often found in patients where the smallest amount of pathological changes are present. The study of this subject suggests that there is still much to be learned about reflexes of the fifth nerve.

Fadism in Medicine — No doctor will be held guiltless who attempts to practice medicine without being familiar with, and willing to utilize, the various approved, auxiliary methods for making a diagnosis.

Likewise, no doctor will be held guiltless who allows his interest in, and enthusiasm for, one special trick to overshadow discretion and blind his perspective. As important as is the test-tube, microscope, and X-ray, a stubborn dependence on their findings alone would often be suicidal and homicidal.

Doctors are human, and it is a trait of humans everywhere to follow fads which often lead to extremes, but certainly no profession and no class of humans need, more than doctors, to sit steady in the boat and make haste slowly.—Editorial, Southern Medicine and Surgery, August, 1922.

PHLORIZIN GLYCOSURIA IN THE DIAGNOSIS OF PREGNANCY *

By MERRILL W. HOLLINGSWORTH, M. D.
(From the Johnston-Wickett Clinic, Anaheim, Calif.)

A review of the attempts to formulate some method of diagnosis of pregnancy by laboratory methods convinces that the necessity for such a test has been felt for some time. Although the menstrual history gives presumptive evidence of the existence or non-existence of pregnancy, we are all familiar with the occasional patient who menstruates several times after becoming pregnant, or even throughout the gestation period. The patient with a history of irregular menstruation before marriage presents an equally perplexing problem. And sometimes the question arises whether the diagnosis should be fibroids alone, pregnancy alone, or fibroids and pregnancy. In such cases a dependable test for pregnancy would be of inestimable value.

Bar and Ecalle, in 1919, named as reactions specific in pregnancy the complement deviation, Abderhalden's dialysis, and the intradermal reactions. To these may be added the epiphanin and cobra-venom reactions and the renal glycosuria, epinephrin glycosuria and phlorizin glycosuria tests.

The laboratory work on which this report is based is limited to the phlorizin glycosuria test, which, like the renal glycosuria and adrenalin glycosuria tests, depends on the occurrence of glycosuria during pregnancy, first noted by Blot, in 1856, later by Duncan, in 1882; the frequency of its occurrence is stated as 86 per cent by Hofbauer, 75 per cent by Payer, 5.75 per cent by Williams, 70 per cent by Stolper, 68 per cent by Berg, and 4 per cent by Cron. Klemperer, in 1896, suggested that the glycosuria of pregnancy may be due to a lowered permeability of the kidney for sugar. Hofbauer, in 1899, considered it due to functional derangement of the liver; in 1911, he undertook to prove this by tests of the liver function and histologic studies. Lenhartz, in 1908, stated that it is relatively harmless. Cristalli, noting that Schroder, Reichenstein, Falk and Hesky, and Bartels had reported a large percentage of levulosuria in pregnancy, supported Hofbauer's hypothesis, as did Sachs, Strauss, and Sebatowski. The investigations of Hynemann, Landsberg, and Heinrichsdorff, however, disproved Hofbauer's hypothesis. They showed that in cases of any derangement of the liver there is some degree of eclamptic toxemia. At this point in the history of the subject blood sugar determinations were applied to the problem. Benthin claimed that the blood sugar during pregnancy is normal or subnormal, a fact corroborated by Novak, Porges and Strisower, Frank, Jacobsen, Mann, and Bergsma. Reichenstein, and later Stolper, considered the glycosuria of pregnancy to be due to a disturbance of ovarian function. The latter undertook to prove his contention by animal experiments, in which he demonstrated that hypo-function

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